IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

APPLICANTS: Cauwenberghs *et al.* CONFIRMATION NO.: 7478

APPLICATION NO.: 10/019,740 GROUP NO.: 1641

FILING DATE: May 8, 2002 EXAMINER: Jung, Unsu

TITLE: Detection of von-Willebrand Factor (vWF) Activity

Mail Stop Amendment
Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

SECOND DECLARATION OF DR. HANS DECKMYN UNDER 37 C.F.R. § 1.132

Dear Sir:

I, Dr. Hans Deckmyn, hereby declare and state as follows:

1. I am a recognized expert in the field of von Willebrand's disease and have been working in this field for many years. My *curriculum vitae*, which includes my educational and employment history and a list of my publications and patents, is attached hereto as Exhibit A. My present position is Professor of Chemistry at KU Leuven Campus Kortrijk. I am also a board member of the Belgian Society on Thrombosis and Haemostasis and an executive officer of the European Thrombosis Research Organization.

2. I am a co-inventor of the subject matter claimed in U.S. Patent Application No. 10/019,740, ("the present application"). I understand that the earliest effective filing date of the present application is July 5, 1999. I am also familiar with the November 17, 2008, Office action as well as the Favaloro, Hoylaerts, and Vischer articles, and the Handin U.S. patent cited therein.

3. von Willebrand's disease (vWD) is a common inherited bleeding disorder associated with defects in von Willebrand factor (vWF), a protein required for platelet adhesion. Type 1 and Type 3 vWD are the result of quantitative defects in vWF production. For example,

Type 1 vWD is characterized by reduced plasma concentrations of vWF as compared to normal, while Type 3 vWD is characterized by negligible to absent vWF concentrations. Type 2 vWD is caused by qualitative defects in vWF, *i.e.*, vWF is functionally abnormal. There are 4 known subtypes of type 2 vWD: 2A, 2B, 2M, and 2N.

4. As of the earliest effective filing date of this application, it was understood by those skilled in the art of vWD that high molecular weight (HMW) multimers of vWF are responsible for inducing platelet aggregation. It was understood that such platelet aggregation is induced when HMW multimers of vWF bind to GP1b receptors on platelets. Further it was understood that multimerization of vWF monomers is necessary for bioactivity of vWF.

5. As of the earliest effective filing date of this application, it was understood by those skilled in the art that each of Type 2A vWD and Type 2B vWD are characterized by the absence of HMW multimers of vWF. In Type 2A vWD patients, the absence of HMW vWF multimers is attributable to errors in protein synthesis that interfere with multimer assembly and/or increased sensitivity of vWF to proteases. As a result, Type 2A patients produce only small fragments or monomers of vWF that cannot multimerize. Consequently, the vWF produced by Type 2A patients has a decreased capacity to interact with platelet GP1b receptor.

6. In contrast, as of the earliest effective filing date of this application, Type 2B vWD was known in the art to be characterized by increased affinity of Type 2B vWF for platelet GP1b. This was known to be the result of a genetic defect that increases the binding affinity of Type 2B vWF for the GP1b receptor. As a result, Type 2B vWF binds to platelet GP1b in circulating blood. Upon binding of vWF to the circulating platelets, platelets aggregate and are cleared from the blood. This accounts for the absence of free (unbound to platelet GP1b) high molecular weight vWF multimers in Type 2B patients. In contrast to Type 2B vWF, normal vWF does not spontaneously interact with the GP1b receptor unless vWF is activated as the result of a bleeding injury.

7. If blood, plasma, or serum from a Type 2B patient sample were incubated with ristocetin and isolated GP1b, a skilled artisan, as of the earliest effective filing date, would have expected to detect only low amounts of vWF binding to isolated GP1b. This is due to the

fact that the HMW vWF multimers would already be bound to GP1b receptors on the patient's own platelets. However, some minimal binding from vWF monomers would contribute to the low amount of vWF binding detected.

8. If blood, plasma, or serum from a Type 2A patient sample were incubated with ristocetin and isolated GP1b, a skilled artisan, as of the earliest effective filing date, would have expected to detect only low amounts of vWF binding to isolated GP1b due to the absence of HMW vWF multimers in the patient sample. However, some minimal binding from vWF monomers would contribute to the low amount of vWF binding. The low level of binding detected between Type 2A vWF and GP1b in the presence of ristocetin discussed here in paragraph 8 would be expected to be the same as the low level of binding detected between Type 2B vWF and GP1b in the presence of ristocetin as discussed in paragraph 7.

9. Based on my statements in paragraphs 4-8 above, as of the earliest effective filing date, a skilled artisan assaying Type 2A or Type 2B patient samples in the presence of ristocetin and isolated GP1b would have expected the results from each assay to exhibit the same level of vWF binding, *i.e.*, a low amount, due to the absence of HMW vWF multimers necessary for binding to GP1b.

10. As of the earliest effective filing date, persons of skill in the art were aware that Type 2A vWD and Type 2B vWD could be distinguished from one another because platelets from a Type 2B patient will aggregate in response to low concentrations of ristocetin, while platelets from a Type 2A patient will not. The difference between platelet aggregation patterns between Type 2A and Type 2B are observed only when patient platelets are assayed and not when exogenous platelets, *i.e.*, non-patient platelets, are assayed in the presence of ristocetin. This assay using patient platelets is known as a ristocetin-induced platelet aggregation (RIPA) assay. The aggregation of Type 2B platelets at low concentrations of ristocetin observed in the RIPA assay is a result of the high affinity for platelet GP1b exhibited by vWF from Type 2B patients. Because of the high affinity for platelet GP1b, Type 2B vWF does not require the same level of cofactor assistance from ristocetin to bind to GP1b as required by normal vWF. In contrast, platelets from a Type 2A vWD patient will exhibit no or a little aggregation in the presence of low or high

concentrations of ristocetin because Type 2A vWF is contains only small vWF monomers which have a low affinity for GP1b, binding to which is necessary to cause platelet aggregation.

11. Based on my statements in paragraphs 4-10, if ristocetin and an isolated GP1b fragment were used in an assay with a patient sample containing patient platelets (patient platelets being required by a RIPA assay), the isolated GP1b would compete with platelet GP1b of patient platelets for binding to defective Type 2B vWF. Given that some defective Type 2B vWF would bind to platelet GP1b and some to isolated GP1b, this would interfere with the results by reducing the expected amount of agglutination. Further, reduced levels of agglutination would also be seen with patient platelets from a Type 2A vWD patient. Because of the reduced levels of agglutination from the expected levels needed to distinguish between Type 2A and Type 2B vWD, it would not be possible to accurately distinguish type 2A and Type 2B vWD from one another employing such a method.

12. As an inventor of the invention presently claimed in this application, I believe that I and the other inventors were the first to recognize the utility of GP1bα isolated from a platelet, as opposed to using whole platelets, in an assay for detecting vWF ristocetin cofactor activity, that is the binding activity of vWF to GP1bα (or a fragment thereof) in the presence of ristocetin or a functionally equivalent substance.

13. In the process of developing a method for discriminating between different types of von Willebrand disease using a vWF binding activity, I and the other inventors first determined whether the binding activity of normal vWF to soluble forms or portions of GP1bα could be adequately detected in the presence of ristocetin by immobilizing the soluble forms or portions of GP1bα to plastic, for example, to 96 well plastic plates, and would therefore be useful for a clinical assay for detecting vWF binding activity in the presence of ristocetin. We assayed both glycocalicin, a naturally occurring soluble fragment of GP1bα, and a recombinant fragment of GP1bα, $GP1b\alpha_{(1-289)}$, for their ability to bind to vWF in the presence of ristocetin. For each GP1bα fragment immobilized to plastic that we tested, we determined that the binding activity of normal vWF in the presence of ristocetin was negligible and therefore not useful for a clinical assay for

detecting vWF binding to GP1bα in the presence of ristocetin. We determined that vWF was unable to bind the immobilized GP1bα fragments because immobilization of the GP1bα fragments causes a conformational change in GP1bα, thereby preventing vWF binding to the GP1bα fragments. Accordingly, we concluded that assaying vWF binding activity to GP1bα or a fragment thereof immobilized to plastic in the presence of ristocetin would not be an effective substitute for detecting vWF ristocetin cofactor activity in the presence of platelet-GP1b because the plastic-immobilized GP1bα fragments did not permit detection of any significant vWF binding activity.

14. Given that immobilizing GP1bα fragments was not successful, we sought other means for utilizing GP1bα fragments in a clinical assay for detecting vWF binding activity in the presence of ristocetin. Subsequently, we discovered that in order to detect the binding activity of vWF to GP1bα or a fragment thereof, it is necessary to present the soluble form or portion of GP1bα by an anti-GP1bα antibody. Over 90 monoclonal antibodies against GP1bα were screened in order to detect anti-GP1bα antibodies, that when bound to $GP1b\alpha_{(1-289)}$, permitted binding of GP1bα to vWF. Over 40 monoclonal anti-GP1bα antibodies were screened in order to detect anti-GP1bα antibodies, that when bound to glycocalicin, permitted binding of glycocalicin to vWF. Anti-GP1bα antibodies were coated on plastic well plates and glycocalicin or a GP1bα fragment were added to the plate. After incubation, normal vWF and ristocetin were added. Binding between vWF and the $GP1b\alpha_{(1-289)}$ fragment or glycocalicin was detected by using an anti-vWF antibody labeled with horseradish peroxidase according to standard ELISA techniques. For 3 anti-GP1bα antibodies, we detected sufficient levels of vWF binding to the GP1bα fragment in the presence of ristocetin. For 1 anti-GP1bα antibody, we detected sufficient levels of vWF binding to glycocalicin in the presence of ristocetin. Accordingly, we determined that these anti-GP1bα antibodies would be suitable for presenting GP1bα in an assay for detecting vWF binding to GP1bα or a fragment thereof in the presence of ristocetin.

15. I further declare that all statements made in this Declaration are of my own knowledge, are true, and that all statements made on information and belief are believed to be true. Moreover, these statements were made with the knowledge that willful false statements

and the like made by me are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issuing thereon.

Dated: May 16, 2007

By: _____

Dr. Hans Deckmyn

Curriculum Vitae Hans DECKMYN

Office address: Laboratory for Thrombosis Research
KU Leuven Campus Kortrijk, E. Sabbelaan 53 B-8500 Kortrijk (Belgium)
tel.: 32-56.246.171 (office), .422 (lab), fax: 32-56.246.997
e-mail: Hans.Deckmyn@kuleuven-kortrijk.be
http://www.kuleuven-kortrijk.be/irc/trombose

Professional positions

1977-1980: Doctoral student Laboratory for Biochemistry (G.Préaux)
1981-1985: Postdoc Center for Thrombosis and Vascular Research, (M. Verstraete, J. Vermylen)
1985-1987: Postdoc Hematology-Oncology, Washington U, School Medicine,St.Louis Mo (P.W. Majerus),
1988-1992: Senior Researcher Center for Molecular & Vascular Biology KU Leuven (D. Collen, J. Vermylen)
1992-1996: Associate Professor in Chemistry KU Leuven Campus Kortrijk
1994-present: Head of the Laboratory for Thrombosis Research, IRC, KU Leuven Campus Kortrijk
1996-1999: Professor in Chemistry KU Leuven Campus Kortrijk
1999-present: Full Professor in Chemistry KU Leuven Campus Kortrijk

Other

1992-1998: Associate Editor "*Thrombosis and Haemostasis*".
1994-1998: Editorial Advisor "*The Biochemical Journal*".
96-02,08-09: Member Research Council KU Leuven
1998-2004: Editor "*The Biochemical Journal*".
1999-2002: Member Advisory Board " *Thrombosis and Haemostasis*".
2000-2006: Chairman Interdisciplinary Research Center (IRC), KU Leuven Campus Kortrijk
2000-2006 Member Board 'Innovation & Incubation Center Kortrijk' (IICK)
2001-2002 Vice-chairman Advisory Council on Innovation Charter Zuid-West-Vlaanderen
2001-2006 Member Board 'Researchpark Hoog Kortrijk'
2002-presentMember Advisory Board " *Journal of Thrombosis and Haemostasis*".
2002-present Member Board 'Belgian Society on Thrombosis and Haemostasis' (BSTH)
2003-presentMember Steering committee 'CIS Innovation West-Vlaanderen'
2004-presentSpokesman "Interfacultary Center for Biomacromolecular Structure Research" (BioMacS)
KU Leuven http://biomacs.kuleuven.be/index.htm
2005-presentBoard member 'European Cardiovascular Genetics Institute' (ECGI)
2005-2007 Member Council for Industrial Research KU Leuven
2006-2008 Section Editor '*Thrombosis and Haemostasis*'
2006-presentMember FWO commission Molecular & Cellular Biology, Genetics

2007-present Executive Officer 'European Thrombosis Research Organisation' (ETRO)
2007-presentFounding member PharmAbs http://www.pharmabs.org/

Various

Honors and awards

1978-1979: Fellowship IWONL (Institute for Advancement of Sc.Res. in Industry & Agriculture, Belgium)
1985: Prize Boehringer-Ingelheim for Research on Thrombosis and Coagulation
1985-1987: NATO Research Fellowship
1985-1986: Fulbright Research Award
1989: Young Investigator Award XIIth Congress on Thrombosis and Haemostasis
1991 Prize "Dr. en Mevr. Schamelhout-Koettlitz "-Foundation for Scientific Research (Royal Academy of Medicine of Belgium)
1992 Fellowship "Belgian Action against Cancer"
1993 Triannual Prize Baron Simonart Foundation for Clinical Pharmacological Research
1995 Research grant O.Dupont Foundation (Royal Academy of Medicine of Belgium)
1997 Visiting professor "Pro Renovanda" University of Debrecen, Hongarije, Medical School
2004 Triannual price *Sidmar* for 'Medical Scientific Research' (Royal Academy for Medicine)
2009 Doctor Honoris Causa, University of Debrecen, Hungary

Memberships, meetings, journals

1. *International Society on Thrombosis and Haemostasis* (ISTH)
   1992-present: Member
   1992-8: Associate Editor Journal of the Society: "*Thrombosis and Haemostasis*"
   1999-2001: Member Advisory Board " *Thrombosis and Haemostasis*".
   2002-present: Member Advisory Board " *Journal on Thrombosis and Haemostasis*".
2. *Biochemical Society*
   1994-present: member
   1994-1998: Editorial Advisor of "The Biochemical Journal"
   1998-2003: Co-Editor of "The Biochemical Journal"
3. *European Thrombosis Research Organisation* (ETRO)
   1995-present: Laboratory for Thrombosis Research, IRC-KULAK elected member
   1997, 2003 Invited speaker ETRO Advanced Teaching Course, Heviz, H; Blankenberge, B

2007-10: Executive Officer & webmaster

4. *Belgian Society on Thrombosis and Haemostasis* (BSTH)
   1993-presentmember
   2002-present member of the board & webmaster
5. *Koninklijke Vlaams Chemische Vereniging*
   1998- present: member

Member Scientific Committee *European Platelet and Granulocyte Immunobiology Symposium*
(Bamberg, D 1992, Cambridge, UK 1994, Hammeenlinna, SF 1996, S'Agaro, E, 1998, Amsterdam,NL, 2000, Lago Maggiore, I, 2002, Rust A, 2004, Tromso, N, 2006, Thun, CH, 2008, Beaune, FR 2010)

Teaching

1992-present: Introductory course 'General Chemistry': 1st yr Medicine, Biomedical Sciences,
Biochemistry, Biology, Chemistry, Physics, Mathematics, Bio-engineers, Pharmacy
1992-2003: Chemistry II (Systematics) 1st yr Biochemistry, Chemistry, Biology

1996-2000: Mechanisms of signal transduction, PhD-training Sci-Bioengin-Pharmacy-Biomedic Sciences
1999-present: Cell Biology (part signal Transduction) : 1st yr Medicine, Biomedical Sciences
2005-present: Biochemistry: 1st yr Biochemistry, Chemistry, Biology, Bioengineers, Pharmacy
Since 2003: promotor 7 PhD thesisses, 5 in preparation

International Advisory functions

1. 1996-present: Dutch "*Nederlandse Hartstichting*"
2. 1997, 2001: Austrian "*Fonds zur Förderung der wissenschaftlichen Forschung*"
3. 1998-present: British "*Medical Research Council*"
4. 1998: South-African "*Medical Research Council*"
5. 1999: *EU-projects* External Expert
6. 99,00, 04: Luxembourg "*Ministère de la Culture, de l'Enseignement Supérieur et de la Recherche*"
7. 2000: Dutch "*Nederlandse Organisatie voor Wetenschappelijk Onderzoek*"
8. 2003: British"*Wellcome Foundation*"
9. 2004: Irish "*Science Foundation*" (SFI)
10. 2004-present French *AERES* (INSERM)

11. 2004: Irish "*Research Funding and Policy Division, Health Research Board* (HRB)"
12. 2004, 2007 : Luxembourg "*Centre de recherche Public de la Santé'*
13. 2006, 2007: French '*Agence Nationale de la Recherche –ANR'* Research on rare diseases
14. 2006: Irish "*Health Research Board*" (HRB)
15. 2006: U of Cambridge UK "*Promotions Committee for the Faculty of Biology*"
16. 2007-present: French"*Agence Nationale de la Recherche–ANR*" 'Pathophysiology of Human Diseases'

At present our research group consists of 2 professors, 4 postdocs, 4 PhD-students and 4 technicians.
The yearly budget (incl. salaries) amounts to ~€ 860.000, all from competitive grants.

Scientific output

Publications

- 175 publications (Medline), 4,806 citations, h-index: 33 (ISI)
of which 1 in Science (26.372), 2 in Circulation (12.755), 24 in Blood (IF 10.896), 7 in ATVB (7.221), 5 in J Thromb Haemost (5.947), 8 in J Biol Chem (5.581),

- 14 chapters in books

Patents

Granted

1. CELL LINES, LIGANDS AND ANTIBODY FRAGMENTS FOR USE IN PHARMACEUTICAL COMPOSITIONS FOR PREVENTING AND TREATING HAEMOSTASIS DISORDERS
European patent granted 2007/01/31, US patent granted
Inventors: N. Cauwenberghs, H. Deckmyn

2. "Inhibition of the vWF-collagen interaction by anti-human vWF monoclonal antibody (82D6A3) results in abolition of in vivo arterial thrombus formation in baboons.
European patent granted 4/2008
Inventors : K. Vanhoorelbeke, N. Cauwenberghs, H. Deckmyn

Pending

1. "Detection of von-Willebrand factor (vWF) activity"
EP patent Application, Inventors: N. Cauwenberghs, K. Vanhoorelbeke, H. Deckmyn

Licenses

A new diagnostic test to determine the quality of von Willebrand Factor, with clearly better reproducibility and sensitivity than the test currently used in the clinic (see patent application and Vanhoorelbeke et al Thromb Haemost. 2000;83:107-13) outlicensed to Instrumentation Laboratories (NJ) and further developed by Biokit (Barcelona). Beta version tested and approved.

Current research interests

1. Development of platelet adhesion inhibitors by interfering with the collagen-VWF-platelet GPIb axis as new antithrombotics with a lower bleeding risk, for treatment of e.g. ischemic stroke or TTP

2. Functional genomics in blood platelets (production and characterisation of platelets from genetically manipulated (silencing/overexpressing) hematopoietic stem cells transplanted in irradiated mice)

Publication list Hans Deckmyn

1. Defreyn G, Deckmyn H, Vermylen J.
A thromboxane synthetase inhibitor reorients endoperoxide metabolism in whole blood towards prostacyclin and prostaglandin E2.
Thromb. Res. 26, 389-400, 1982

2. Badenhorst PN, Deckmyn H, Vermylen J.
The effect of sulphinpyrazone on whole blood thromboxane and prostacyclin generation in man.
Thromb. Res. 28, 59-66, 1982.

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3. Boogaerts MA, Vermylen J, Deckmyn H, Roelant C, Verwilghen RL, Jacobs HS, Moldow CF.
Enkephalins modify granulocyte-endothelial interactions by stimulating prostacyclin production.
Thromb. Haemost.50, 572-575, 1983.

4. Deckmyn H, Proesmans W, Vermylen J.
Prostacyclin production by whole blood from children: impairment in the hemolytic uremic syndrome and excessive formation in chronic renal failure.
Thromb. Res. 30, 13-18, 1983.

5. Spitz B, Deckmyn H, Van Assche FA, Vermylen J.
Prostacyclin production in whole blood throughout normal pregnancy.
Clin.Exp.Hypert.-Hypert. in Pregnancy B2, 191-202, 1983.

6. Vermylen J, Badenhorst PN, Deckmyn H, Arnout J.
Normal mechanisms of platelet function.
Clin. Haematol. 12,107-151, 1983.

7. Vermylen J, Deckmyn H.
Reorientation of prostaglandin endoperoxide metabolism by a thromboxane synthetase inhibitor: in vitro and clinical observations.
Br.J.Clin.Pharmacol. 15, 17S-22S, 1983.

8. Deckmyn H, Font L, Van Hemelen C, Carreras LO, Defreyn G, Vermylen J.
Low prostacyclin synthetase activity of fetal rat aorta.
Life Sci. 33, 1491-1497, 1983.

9. Deckmyn H, Van Houtte E, Verstraete M, Vermylen J.
Manipulation of the local thromboxane and prostacyclin balance in vivo by the antithrombotic compounds dazoxiben, acetylsalicylic acid and nafazatrom.
Biochem. Pharmacol.32, 2757-2762, 1983.

10. Gresele P, Zoja C, Deckmyn H, Arnout J, Vermylen J, Verstraete M.
Dipyridamole inhibits platelet aggregation in whole blood.
Thromb. Haemost. 50, 852-856, 1983.

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11. Van Assche FA, Spitz B, Vermylen J, Deckmyn H.
Preliminary observations on treatment of pregnancy induced hypertension with a thromboxane synthetase inhibitor.
Am. J. Obstet. Gynecol. 148, 216-218,1984.

12. Boogaerts MA, Van de Broeck J, Deckmyn H, Roelant C, Vermylen J, Verwilghen RL. Protective effect of vitamin E on immune triggered granulocyte mediated endothelial injury.

Thromb. Haemost. 51, 89-92, 1984.

13. Deckmyn H, Gresele P, Arnout J, Vermylen J.
BM 13.177 specifically blocks the platelet thromboxane receptor.
Arch. Intern. Pharmacodynam. Ther. 268, 165-166, 1984.

14. Essien EM, Arnout J, Deckmyn H, Vermylen J, Verstraete M.
Blood changes and enhanced thromboxane and 6-keto prostaglandin $F_{1\alpha}$ production in experimental acute plasmodium bergei infection in hamsters.
Thromb. Haemost. 51, 362-365, 1984.

15. Gresele P, Deckmyn H, Arnout J, Vermylen J.
Platelet inhibitory activity of prostacyclin in the presence of erythrocytes as studied with the impedance aggregometer.
Br. J. Haemat. 57, 171-173, 1984.

16. Gresele P, Deckmyn H, Huybrechts E, Vermylen J.
Serum albumin enhances the impairment of platelet aggregation with thromboxane synthase inhibition by increasing the formation of prostaglandin D_2.
Biochem. Pharmacol. 33, 2083-2088, 1984.

17. Gresele P, Deckmyn H, Arnout J, Lemmens J, Janssens W, Vermylen J.
BM 13.177, a selective blocker of platelet and vessel wall thromboxane receptors, is active in man.
Lancet i, 991-994, 1984.

18. Deckmyn H, Gresele P, Arnout J, Todisco A, Vermylen J.
Prolonging prostacyclin production by nafazatrom and dipyridamole.
Lancet ii, 410-411, 1984.

19. Spitz B, Deckmyn H, Van Assche FA, Vermylen J.
Prostacyclin in pregnancy.
Eur. J. Obstet. Reprod. Biol. 18, 303-308, 1984.

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20. Arnout J, Vanrusselt M, Deckmyn H, Vermylen J, Fiocchi R, Lijnen P, Amery A.
Platelet hypersensitivity to serotonin after prolonged ketanserin intake?
J. Cardiovasc. Pharmacol. 7 (suppl.7), S20-S22, 1985.

21. Ceuppens JL, Vertessen S, Deckmyn H, Vermylen J.
Effects of thromboxane A_2 on lymphocyte proliferation.
Cell. Immunol. 90, 458-463, 1985.

22. De Maeyer P, Deckmyn H, Arnout J, Vermylen J.
Intravenous ionic contrast media cause local prostacyclin release in man
Investigative Radiol. 20, 458-463, 1985.

23. Deckmyn H, Zoja C, Arnout J, Todisco A, D'Hondt J, Vanden Bulcke F, Hendrickx N, Gresele P, Vermylen J. Partial isolation and function of the prostacyclin regulating plasma factor.
Clin. Sc. 69, 383-393, 1985.

24. Gresele P, Arnout J, Deckmyn H, Vermylen J.
Combining antiplatelet agents: potentiation between aspirin and dipyridamole.
Lancet i, 937-938, 1985.

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Thromboxane A_2 and prostacyclin do not modulate the systemic hemodynamic response to cold in humans.
J. Lab. Clin. Med. 106, 534-541, 1985.

26. Janssens WJ, Deckmyn H, Gresele P, Vermylen J.
BM 13.177 selectively inhibits endoperoxide analog induced vascular contractions.
Arch. Intn'l Pharmacodyn. Ther. 276, 28-34, 1985.

27. Spitz B, Deckmyn H, Van Bree R, Pijnenborg R, Vermylen J, Van Assche FA.
Influence of a vitamin E deficient diet on prostacyclin production by mesometrial triangles and aortic rings from non-diabetic and diabetic rats.
Am. J. Obstet. Gynecol. 151, 116-120, 1985.

28. Van Renterghem Y, Roels L, Lerut T, Gruwez J, Michielsen P, Gresele P, Deckmyn H, Colucci, M, Arnout J, Vermylen J.
Thrombo-embolic complications and haemostatic changes in cyclosporin-treated cadaveric kidney transplants.
Lancet i, 999-1002, 1985.

29. Gresele P, Deckmyn H, Arnout J, Zoja C, Vermylen J.
Lack of synergism between dazoxiben and dipyridamole following administration to man.
Thromb. Res. 37, 231-236, 1985.

30. Vermylen J, Deckmyn H, Arnout J, Gresele P.
A. Schmidt Memorial Lecture: Peroxides in haemostasis and thrombosis.
Haemostasis 15, 8-9, 1985.

31. Vervliet G, Deckmyn H, Carton H, Billiau A.
Influence of prostaglandin E_2 and indomethacin on interferon-gamma production by cultured peripheral blood leukocytes of multiple sclerosis patients and healthy donors.
J. Clin. Immunol. 5, 102-108, 1985.

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32. Gresele P, Arnout J, Deckmyn H, Vermylen J.
Mechanism of the antiplatelet action of dipyridamole in whole blood: modulation of adenosine concentration and activity.
Thromb. Haemost. 55, 12-18, 1986.

33. Vermylen J, Arnout J, Deckmyn H, Xhonneux B, De Clerck F.
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Thromb. Res. 42, 721-723, 1986.

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Thrombosis and immune disorders.
Clin. Haematol. 15, 393-412, 1986.

35. Gresele P, Arnout J, Coene MC, Deckmyn H, Vermylen J.
Leukotriene B_4 production by stimulated whole blood: comparative studies with isolated polymorphonuclear cells.
Biochem. Biophys. Res. Commun. 137, 334-342, 1986.

36. Majerus PW, Connolly TM, Deckmyn H, Ross TS, Bross TE, Ishii H, Bansal V, Wilson DB. The production of phosphoinositide-derived messenger molecules.
Science 234, 1519-1526, 1986.

37. Deckmyn H, Tu SM, Majerus PW.
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J. Biol. Chem. 261, 16553-16558, 1986.

38. Ceuppens JL, Robaeys G, Verdickt W, Vertessen S, Deckmyn H, Dequeker J.
Immunomodulatory effects of non-steroidal anti-inflammatory agents in vivo on lymphocyte function in vitro.
Arthritis Rheumat. 29, 305-311, 1986.

39. Kienast J, Arnout J, Deckmyn H, Pfliegler G, Hoet B, Vermylen J.
On the role of guanine nucleotide binding regulatory proteins (G-proteins) in signal transduction in human platelets: studies with sodium fluoride (NaF).
Agents Actions, Suppl. 20, 175-180, 1986.

40. Kienast J, Arnout J, Pfliegler G, Deckmyn H, Hoet B, Vermylen J.
Sodium fluoride mimics effects of both agonists and antagonists on intact human platelets by simultaneous modulation of phospholipase C and adenylate cyclase activity.
Blood 69, 859-866, 1987.

41. Gresele P, Arnout J, Deckmyn H, Huybrechts E, Pieters G, Vermylen J.
Role of proaggregatory and antiaggregatory prostaglandins in hemostasis. Studies with combined thromboxane synthase inhibition and thromboxane receptor antagonism.
J. Clin. Invest. 80, 1435-1445, 1987.

42. Arnout J, Kienast J, Deckmyn H, Vermylen J.
Prostacyclin stimulatory activity of reducing cofactors in human plasma filtrate. A potential role for uric acid.
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